SPASTIC DYSPHONIA
1871-1990

Kaori Miyatake
Rakuno Gakuen University

ABSTRACT

現在 Spasmodic Dysphonia という言葉で統一されている痙攣性発声障害に関する文献が初めて世の中に出たのは 1871 年である。これまでに様々な分野からこの音声障害が調査研究されているが、長らくその病因も病態もはっきり特定することが出来ていない。その理由の一つに患者数が稀でその全体像が掴めていないことがあげられる。ここではまず時代を区切って、Traube の文献から 1990 年までの文献をまとめる。1991 年以降は改めて述べる機会を持ちたい。

INTRODUCTION

Spastic dysphonia is a controversial voice disorder. Since 1871, when Traube first described it as "a spastic form of nervous hoarseness", spastic dysphonia has undergone historical changes of its definition.

Initially, it was believed that spastic dysphonia was a psychogenic disorder. Arnold (1959) noted that it was caused by "psychoneurosis from either occupational stress or emotional trauma, such as family conflicts, accidents, terrifying events or accumulated frustration." Robe, Brumlik and Moore (1960) disagreed and reported that spastic dysphonia was a disease of the central nervous system. Followed by the evolution of the field of neurology, a neurologic etiology has been suggested with a variety of potential lesions: periphery (Bocchino & Tucker, 1978; Dedo, Townsend & Izdebski, 1978), brain stem or medulla (Kiml, 1965; Hall, 1981), extrapyramidal system or basal ganglia (Critchley, 1939; Aronson, Brown, Litin & Pearson, 1968; McCall, 1974; Aminoff, Dedo & Izdebski, 1978; Jankovic & Ford, 1982), and cerebral cortex (Robe, Brumlik & Moore, 1960; Maroun, Jacob & Gowing, 1970). Blitzer and his colleagues (1985) classified spastic dysphonia as focal laryngeal dystonia, a disorder of central motor processing. Although many cases of spastic dysphonia have been found to be of neurologic etiology, there are other cases for which either psychogenic or unknown etiologies remain. There are wide fluctuations in severity of spastic dysphonia. Many researchers have reported that emotional stress makes the disorder worse. In addition to these unresolved etiologic issues, the name "spastic" itself is termed a misnomer since it is not related at all to spastic paralysis (Bloodstein, 1984). Aminoff (1979) and others, thus, suggested the label "spasmodic dysphonia" rather than "spastic dysphonia".

WHAT IS SPASTIC DYSPHONIA?

Symptoms

Imagine having to speak while also trying to lift a car by yourself. The resulting voice is one might expect from the patient with spastic dysphonia.

The Vocal Characteristics of Spastic Dysphonia include: tension, strain, breathiness, overpressure, effortful, jerky, staccato, hoarse, spastic, stuttering-like, hyperaddution tremor, grunts, groaning, laborious, strangled, creaking, squeezed, chocked vocal attack, periodic breaks, stoppages, pinched, grating, uncontrolled pitch and loudness (a tendency to be monopitched and reduced in loudness), hostile-sounding, and vowels that are initiated with hard glottal attack and voice arrests (Arnold, 1959; Luchsinger & Arnold, 1965; Dedo & Shipp, 1980; Izdebski & Dedo, 1981; Aronson, 1985). "This abnormal voice occurs only during voluntary phonation for communication purposes" (Aronson, 1985, p.157). It may be most severe when the patient needs to interact with other people. Dedo (1980) reported that the telephone conversation was the most universally difficult task for the patients than any other setting. However, patients can make normal phonation during laughter, anger, singing, shouting, talking aloud to her/himself, saying "ee" in a high pitched voice (vowel prolongation) or after drinking alcohol or while emerging from general anesthesia (Dedo & Shipp, 1980; Boone, 1983; Stemple, 1984; Aronson, 1985). These symptoms are reminiscent of stuttering. There is wide fluctuation of symptoms within stuttering and within spastic dysphonia, based on how severe the disorder is and the detailed voice characteristics (Aronson, 1985).

Pathophysiology

The universal denominator is adductor laryngospasm. Aronson described in detail what happens to the vocal folds and muscles of the larynx during the spasms:

1. In a mild case, hyperadduction (adductor spasms) of the true vocal fold alone occurs. "The vocal folds can be seen to snap shut synchronous with each voice arrest."
2. In a moderate to sever case, "adductor spasms of both the true and false vocal folds occur. The false or ventricular vocal folds close over and obscure the true folds."
3. In a severe case, supraglottic constriction occurs, which means "severe strained voice or voice arrests are accompanied by constriction of the hypopharynx, i.e., the inferior pharyngeal constrictor just above the level of the false vocal folds" (Aronson, 1985, p.159).

Aronson (1985) reported that "the more severe the adductor laryngospasms, the greater the vertical extent of intrinsic laryngospasms, the greater the vertical extent of intrinsic laryngeal muscle participation. As tightness of spasms increase, constriction progresses superiorly from the true folds to involve the ventricular folds and, ultimately, the pharyngeal constrictors" (p. 159). Synchronous movements of the entire larynx occur in a superior direction along with the moderate to severe laryngospasms. The extrinsic laryngeal muscles also move together, and so does the skin of the neck. The Xth nerve to the intrinsic laryngeal muscles, the IXth nerve to the pharynx, and the cervical spinal nerves to the extrinsic laryngeal muscles are involved with the spasms. Dedo and Shipp (1980) cited four specific muscles' failure to effect precise contraction: the posterior cricoarytenoid, the thyroarytenoid, the lateral cricoarytenoid and the interary-

tenoid. Spastic dysphonia can be said to be a disorder of not only the true and false vocal folds, but also the supraglottic pharyngeal constrictors and the strap muscles (Aronson, 1985).

Accompanying Disorders

The laryngospasm of spastic dysphonia is frequently accompanied by other disorders, e.g. vocal tremors, abnormal respiratory movements ("probably the secondary effect of the uncontrolled glottic closures", Aronson, 1985, p.160), dysrhythmic movements of the thorax and abdomen, head jerking, and eye blinking (Dedo & Shipp, 1980; Stemple, 1984; Aronson, 1985). Stemple (1984) reported complaints, such as physical fatigue, tightness of the chest, back, and shoulder muscles, and shortness of breath caused by the efforts to phonate through the tight glottal closure. As a compensation, patients with spastic dysphonia often speak on inhalation, whisper, and use many facial grimaces of the eyes, cheeks, nose, lips and jaw. There is a tendency to alter the articulation pattern, such as a substitution of voiced for unvoiced sounds (Dedo& Shipp, 1980). These behaviors resemble those of stuttering; interestingly, Dedo and Shipp (1980) provided data that some patients had histories of stuttering prior to the spastic dysphonia. Some patients demonstrated obvious central nervous problems, such as torticollis, mild cerebral palsy, or fine tremors involving one or both arms and legs (Dedo & Shipp, 1980).

DATA ON SPASTIC DYSPHONIA

Incidence and Sex Ratio

The incidence and prevalence of spastic dysphonia is still unknown. Many doctors, speech pathologists and researchers mention that it is rare to have a patient with spastic dysphonia. The lack of data limits the ability to identify the specific etiology, and the unsettled identification of the accurate etiology also prevents collecting the precise data to find out incidence and prevalence. The limitation of the data on spastic dysphonia is shown in Table 1 (Dedo & Shipp, 1980; Aronson, 1985).

Table 1
Sex Ratio of Patients with Spastic Dysphonia

Source	# of Cases	Ratio (M:F)	Reference
Bauer (Heidelberg)	19	1:1.7	Kiml (1963)
Brodnitz (USA)	34	1:1.4	Kiml (1963)
Fritzell (Goteborg)	3	1:0.5	Kiml (1963)
Kiml (Praha)	8	1:1	Kiml (1963)
Perello (Barcelona)	8	1:1	Kiml (1963)
Van Thall (London)	6	1:2	Kiml (1963)
Robe et al. (USA)	10	1:4	Robe et al. (1960)
Aronson et al. (USA)	34	1:1.4	Aronson et al. (1968)
Brodnitz (USA)	130	1:1.3	Brodnitz (1976)
Aronson (USA)	100	1:2	Aronson (1980)
Dedo et al. (USA)	- - -	1:2	Dedo et al. (1980)
Izdebski et al. (USA)	200	1:2.3	Izdebski et al. (1984)

Age of Onset and Course

Case (1984) noted that spastic dysphonia was an adult disorder. The average age of onset in spastic dysphonia is about 50 years old, however there is a wide range of duration of symptom awareness before consultation. Aronson (1985) reported an average 8 years interval between the real onset and subsequent consultation. An unusual case was reported by Dedo and Shipp (1980), of a 79 year-old woman who had experienced spastic dysphonia for 72 years before seeking help, which means a possible onset at the age of seven years old. Table 2 summarizes several studies reported by researchers. "Severity may plateau at any point between a mild to severe spasticity with the median time period from onset to the final plateau being approximately 1 year" (Stemple, 1984, p.78).

Factors Associated with Onset

Boone (1983) wrote that spastic dysphonia was affected by the difficulties in interpersonal adjustment. As many researchers have discussed in the literature, acute or chronic psychological stress is associated with this disorder. However, none of the literature can specify stress as whether a primary cause or a trigger incident of this disorder. If stress were a primary cause, spastic dysphonia would be a considered psychogenic. On the other hand, if stress was a trigger, spastic dysphonia could be described as a latent neurologic disorder. Similar to the psychogenic voice disorders as a group, spastic dysphonia is commonly reported to occur shortly after upper respiratory infections, such as a cold, sore throat, or laryngitis. Nevertheless, Aronson (1985) questions whether the illnesses were true physical infections or whether they were unconscious simulations by the psychological rationale for announcing the disorder.

Table 2
Percent of Age Range of Spastic Dysphonia

Source	# of Patient	Range of Age	Mean Age of Onset	% of Age Range
Aronson (1968)	34	28-69	44	20-29 9% 30-39 24% 40-49 29% 50-59 20% 60-69 18%
Brodnitz (1976)	130	27-76	50.2	20-30 10% 31-40 16% 41-50 27% 51-60 29% over 60 18%
Aronson (1979)	100	22-77 (F) 21-72 (M)	50 (F) 48 (M)	21-29 9% 30-39 11% 40-49 29% 50-59 37% 60-69 8% 70-79 6%
Dedo & Shipp (1980)	- - -	17-92	62	- - - - - - - - - -
Finitzo et al. (1989)	75	20-71	- -	- - - - - - - - - -

Psychosocial Effects

Regardless of the etiology, the voice disorder itself affects the patient's equilibrium psychologically. Aronson (1985) described the patient's psychological tendencies: "Self-consciousness, feelings of inadequacy, paranoid ideation, diminished interpersonal communication, social withdrawal, alcoholism, depression, and even suicidal tendency" (Aronson, 1985, p.165). It is well known that losing one's normal voice damages not only the individual's mental health, but also her/his social and occupational life. The degree of the disadvantages caused by spastic dysphonia depends on each patient in terms of personality and life circumstances. It is easy to imagine the more she or he needs to talk at work, the more she or he finds occupational disability. Some are forced to change jobs.

Factors for Severity

It has already been discussed that the patient with spastic dysphonia experiences wide fluctuations in severity. Just like stuttering, the severity fluctuates under different conditions: emotionally, physically, and situationally. Feeling relaxed and free from anxiety makes one's voice better or normal. Aronson (1985) reported that going on vacation made the patient's voice better. Spontaneous speech and a pleasant thought and mood can make the patient speak in normal voice. Aronson (1985) concluded that "spasms are linked to volitional, intellectual, or executive speech functions and disappear during uninhibited, emotional, automatic, nonintellectual speech" (p.164).

ETIOLOGY

Psychogenic

Aronson (1985) suggested that the diagnosis of spastic dysphonia as a psychogenic disorder be made if the onset of the disorder and a stressful life event appeared related to each other. Significant improvements in vocal quality could appear during the revelation of confidential information to the clinician, or during trial symptomatic voice therapy, or while on vacation. Organic and neurologic voice disorders do not have these extreme fluctuations.

In psychogenic spastic dysphonia, there are two types of reactions: conversion reaction and musculoskeletal tension reaction.

1) **Conversion Reaction** (Block, 1965; Kiml 1963): "spastic dysphonia serves to erect a barricade against the dangers of self-revelation. A regressive phenomenon parallel to primitive laryngeal reflex, such as coughing, gagging, wretching, vomiting, and swallowing" (Aronson, 1985, p.169). Heaver (1959) and Aronson (1985) provided several cases of this conversion reaction, and Aronson concluded it was "common in the conversion reaction type to have hostility, verging on rage, inability to verbalize aggression, and entrapment in life circumstances with no apparent means of escape" (p.174). The psychiatric follow-up revealed further evidence that some spastic dysphonia patients had psychoneurotic histories. Personality characteristics include tendencies toward perfectionism, anger suppression, and verbal repression.

2) **Musculoskeletal Tension Reaction** (Aronson, 1985): Although this type of dysphonia is caused by heightened anxiety, tension or depression, and labeled as psychogenic, it is no more

serious than the conversion reaction type. Musculoskeletal tension is simply a muscle tension reaction, which results in vocal hyperfunction.

Neurologic

It is almost impossible to distinguish the neurologic spastic dysphonia from the psychogenic spastic dysphonia superficially without clear neurologic signs. Even the neurologists would have a difficult time determining the difference. Aronson (1985) defined the lesion/lesions within the extrapyramidal system, which occurred singly or in combination. The research done by Finitzo and Freeman (1989) reported that spastic dysphonia was "a supranuclear movement disorder primarily, but not exclusively, affecting the larynx" (1980, p.553), and provided data that half of their subjects showed isolated functional cortical lesions. They concluded that one form of spastic dysphonia reflected "combinations of focal cortical dysfunction-specifically, left frontal/temporal cortex, paramedian frontal cortex, and right posterior temporal/parietal cortex" (1989, p.553). Dedo, Townsend, and Izdebski (1978) provided data of signs of neurologic disturbances: postural tremor, blepharospasm, idiopathic torsion dystonia, and buccolingual dyskinesia. Dedo did further histologic examinations of recurrent laryngeal nerves that were excised from the patients, and concluded 30% demonstrated significant structural differences from normal ones. Hartman (1980) found that limb tremor was the most prominent sign of neurologic disturbance. Aronson (1985) classified 2 types of neurologic spastic dysphonia. One type is organic (essential) voice tremor, which is a component of the essential voice tremor, best demonstrated during vowel prolongation, involves the larynx only, and is produced by alternating adduction and abduction of the true vocal folds and, often, false vocal folds (which may be synchronous with vertical oscillations of the larynx). Organic voice tremor can be confirmed by rhythmic, semirhythmic, or irregular voice arrests during vowel /a/ prolongations. Emotional stress can be a precipitant of organic voice tremor too. Organic voice tremor cannot be qualitatively different from the psychogenic type. Death of a relative or tragedies in the family are common for both the etiology of psychogenic and/or the trigger of neurologic type. The other type of neurologic spastic dysphonia can also be a laryngeal sign of dystonia. In these syndromes, Aronson (1985) included Meige's syndrome, which is the medial facial spasm, and spasmodic torticollis as an uncontrollable pulling, twisting, or tilting of the head to one side. Aronson and DeSanto (1983) stated, additionally, that orofaciallaryngeal dyskinesia and chorea were also responsible for laryngeal spasm. Another approach to identification of spastic dysphonia has been done by McCall (1973), Hall and Jerger (1976), Schaefer, Finitzo-Hieber, Gerling, and Freeman (1983). According to Hall (1981), a subtle central auditory impairment at the brain stem level might exist in the patients with spastic dysphonia. Dedo and other researchers identified spastic dysphonia as a peripheral nervous system lesion, specifically of the Xth nerve and was of viral origin (Dedo et al., 1978; Bocchino & tucker, 1978).

Idiopathic

There were patients in whom no convincing evidence of psychogenic or neurologic etiology can be found. Since Aronson noted many times that the term "spastic dysphonia" has been used to "describe the voice symptom, disregarding the possibility that they might have been dealing

with a spastic dysphonia of a different species" (1985, p.168), it is understandable that there are unknown etiologies among the spastic dysphonia patients.

DIAGNOSIS

Aronson (1990) noted "therapy is more often successful when it is based on a solid foundation of diagnostic examinations" (p.334). The differential diagnosis by different disciplines, such as the speech pathologist, laryngologist, neurologist, psychiatrist, psychologist or psychiatric social worker, can narrow down the etiology and treatment thereby.

Laryngologic Examination by the Otolaryngologist

1. This examination has to be done in order to clear the doubt of tumors or other structural lesions. The patients often hold of tumors or other structural lesions. The patients often hold that fear. 2. Vocal nodules, polyps, or contact ulcers may contribute to the severity of the dysphonia. 3. Laryngoscope examination to look at rhythmic adduction of the vocal folds producing voice tremor and also rhythmic hyperaddution producing voice arrest may be important diagnostically. Dedo (1980) suggested the use of audiorecording to obtain the patient's vocal behavior with name, address, phone number, counting from one through ten, and using the same demonstration holding a telephone, since this task may make the voice worse.

Speech Examination

A tape recorder with the best recording equipment available is vital for evaluation and comparison during and after therapy. Dedo (1980) suggested use of Fairbanks' Rainbow Passage and sustaining the vowel /a/ in three conditions: a comfortable pitch level as long as possible, a high falsetto briefly, and the lowest modal frequencies possible. Aronson (1985) proposed inclusion of a motor speech test for dysarthria. He also noted "the musculoskeletal tension testing and brief efforts to reduce such tension through laryngeal manipulation" (p.365).

Psychosocial Examination

The patient frequently demonstrates better or normal voice while revealing her/his psychodynamic information related to the voice disorder.

Neurologic Examination

When tremor or movement disorders are suspected to be related to spastic dysphonia, further neurologic examination is needed.

TREATMENT

For years, many modification approaches including voice therapy, drug therapy, psychotherapy, hypnosis, acupuncture, relaxation therapy, and biofeedback training have been done and have not worked well. Aronson (1985) reviewed some of the treatments, which have been at least partially successful.

Symptomatic Voice Therapy

This therapy would be used in the first stage of the treatment unless the neurologic type of spastic dysphonia has been definitely established.

1) **Musculoskeletal Tension Reduction**

This facilitator can often bring normal or near normal voice within a brief period of time if spastic dysphonia is non organic.

> Aronson (1990) explained the technique of musculoskeletal tension reduction: 1) Encircle the hyoid bone with the thumb and middle finger, working them posteriorly until the tips of the major horns are felt. 2) Exert light pressure with the fingers in a circular motion over the tips of the hyoid bone and ask if the patient feels pain, not just pressure. It is important to watch facial expression for signs of discomfort or pain. 3) Repeat this procedure with the fingers in the thyrohyoid space, beginning from the thyroid notch and working posteriorly. 4) Find the posterior borders of the thyroid cartilage just medial to the sternocleidomastoid muscles and repeat the procedure. 5) With the fingers over the superior borders of the thyroid cartilage, begin to work the larynx gently downward, also moving it laterally at times. One should check for a lower laryngeal position by estimating the increased size of the thyrohyoid space. 6) Ask the patient to prolong vowels during these procedures, noting changes in quality or pitch. Clearer voice quality and lower pitch indicate relief of tension. Because these procedures are fatiguing, rest periods should be provided. 7) Once a voice change has taken place, the patient should be allowed to experiment with the voice, repeating vowels, words, and sentences (pp.314-315).

2) **Voice Quality and Pitch Modification**: Except for the essential tremor type of spastic dysphonia, the patient can usually phonate more easily at a higher pitch or by using a breathy voice, which means she or he may be able to sustain spasm-free voice even during daily conversational speech. Cooper (1980) established a therapy program with the belief that voluntary alteration of phonation and respiration can benefit the patient with spastic dysphonia.

Cooper's therapy program is addressed: 1) re-establishment of natural or optimum pitch and "um-hum" method, 2) correct breath support using gentle, abdominal breathing, 3) "peripatetic" voice therapy during which the clinician works with the patient at the actual site and under actual speaking conditions (Aronson, 1990, p.336; Cooper, 1980).

Psychotherapy

Historically psychotherapy has been little used, however, it is definitely beneficial for the patients with spastic dysphonia. Rollin (1987) addressed that regardless of etiology or predisposition, the distress caused by spastic dysphonia may have a profound effect upon interpersonal relations. Therefore, anger, depression, withdrawal, and in extreme cases, definitive personality changes may occur. In terms of differential diagnosis, spastic dysphonia due to conversion reaction or to psychogenic may yield better or normal voice.

Surgical Treatment

1) **History**

In 1952, Rethi was the first person to perform resection of the stylopharyngeus muscle to relieve sphincter laryngospasm of a spastic-like disorder. Twenty years later, Hannebert (1972) supported injection to either the posterior wall of the pharynx or the stylopharyngeus muscle. He believed the effect of the anesthetized sphincters would make the voice better. However, neither surgery nor injection ever became popular as a therapy for spastic dysphonia. When Dedo first introduced the recurrent laryngeal nerve section in 1976, that was the real dawn of the long-term treatment of spastic dysphonia. Followers have done this surgery in their own ways, but Dedo's section will be emphasized in this paper.

2) **Dedo's Recurrent Laryngeal Nerve Resection**

Prior to the resection, the evaluation of the surgical candidate is performed by the speech pathologist, the otolaryngologist, and the surgeon in order to make the final decision for the operation. The procedure of the evaluation follows essentially the same sequence as outlined in this paper (Diagnosis, Laryngologic Examination, Speech Examination). In addition to these evaluations, a chemical paralysis of the vocal folds is performed. Dedo (1980) mentioned that it is the custom to block the recurrent nerve on one side with lidocaine 1% in order to give the patient a preview of the post-surgical voice. This preview lasts approximately 3 to 20 minutes. During this state, the patient undergoes speech therapy instruction to practice controlling the altered vocal structures. This is a very good opportunity for the candidate to make up her/his mind as to whether to undergo this operation. Surgery could result in the patient's laryngeal strain, and this fascinates the patient more than the improved voice. The more positive the patient feels about the operation, the more likely the surgery will be successful. The highly motivated is more likely to recover her/his normal voice. At the same time, the patient needs to undergo counseling. The surgeon cannot guarantee a normal voice, and the patient needs to understand this. The patient is not used to the new, post-surgical voice. The patient might have a negative image of the new "nearly normal voice", which appears after all these years. The family members or friends of the patient are strongly advised to encourage the patient. In this way, these people become a part of the treatment group too. Dedo (1980) reported that he typically saw patient the night before the surgery to explain the usual course of events for the following days. Dedo advised the patient to use the voice as much as desired, because use of the voice is not harmful, even on the first day. The patient is also informed how the surgery is done.

Surgical Treatment

The following is the explanation given to the patients before the patients before the operation (Dedo & Shipp, 1980).

> A horizontal incision approximately 2 inches long in the lower anterior neck just above the manubrial notch, and below the inferior edge of the cricoid cartilage, which leaves a minor scar by plastic surgical techniques, is made. First, identifying the recurrent nerve and removing a segment after confirming its location by electrical stimulation with observation of the vocal fold through a laryngoscope.

> Sectioning the recurrent nerve paralyzes four of the five muscles controlling the position of one vocal fold. This causes the paralyzed vocal fold to stop opening on inspiration and closing to the midline during phonation and swallowing. Instead, the paralyzed fold remains in the paramedian position approximately 1mm to 2mm back from the midline. This position results in less-firm closure with the nonparalyzed fold during phonation and deglutition (p.26).

Recurrent Nerve Section

> "Under general anesthesia a collar incision is made a finger-breadth below the cricoid cartilage. Dissection is carried down to the anterior surface of the trachea and around its right side to the tracheoesophageal groove. The recurrent nerve is identified ordinarily at the level of the inferior pole of the right thyroid gland 1cm. lateral to the trachea . . . Direct laryngoscope is then performed so that the right vocal cord can be observed while an assistant crushes the presumed recurrent nerve with a hemostat. If the vocal cord contracts abruptly at the instant of crushing, the structure is presumed to be the whole recurrent nerve and is ligated . . . A 1cm. segment is then removed adjacent to the inferior pole of the thyroid gland about 3-4cm. below the cricoid (Dedo, 1976, pp.454-455).

Dedo usually sections the left side of the recurrent nerve because the longer left nerve is more vulnerable to trauma throughout the patient's post-operative lifetime. Resection of the superior laryngeal nerve and the recurrent laryngeal nerve crush was considered, but after the experiments Dedo and his associates concluded that recurrent nerve section only as an effective treatment.

Post-Operative Counseling

For the following reason, it is important to see the client the evening of the surgical day to tell her or him that these disturbing realizations are within the normal stages: 1) pain may occur not only in and around the incision area, but also in the region of the posterior neck and chest, 2) swallowing of liquids is sometimes troublesome, as some branches of the recurrent laryngeal nerve innervate musculature in the laryngeal vestibule, which acts to protect the airway from intrusion during swallowing, and 3) coughing is not efficient since the surgery altered the mechanism for tight laryngeal closure. At this stage, the voice may vary from normal to very distorted, grossly aperiodic, or even aphonic (Dedo & Shipp, 1980).

Post-Operative Voice Quality Characteristics

The first day after the operation, if the patient can sustain the vowel /u/ while changing the vocal frequency from the highest falsetto to the lowest sustainable frequency with an easy, comfortable effort and level clearly without breaks, it is a very optimistic sign (Dedo & Shipp, 1980). Only a few patients who underwent the surgery could obtain normal voice and the rest showed some type of aberrant voice quality, which needed speech therapy afterwards. The common difficulties are low-intensity, low-pitch monotonicity, aperiodicity, breathiness, diplophonia, voice tremor, and vocal fry. Even though the early voice tends to be breathy, weak and hoarse, it will become clearer and louder in most cases (Aronson, 1985).

Post-Operative Anatomy

After the recurrent laryngeal nerve section, there is a unilateral paralysis of 4 out of 5 muscles responsible for positioning that one vocal fold. Dedo and Shipp (1980, p.33) described them: 1) the thyroarytenoid muscle that makes up the body or the greater bulk of the vocal fold itself, 2) the posterior cricoarytenoid muscle, which is the primary vocal fold abductor, 3) the interarytenoid muscle that acts reciprocally with the posterior cricoarytenoid to position the vocal fold toward the midline, and 4) the lateral cricoarytenoid, also a muscle of vocal fold adduction. The cricothyroid muscle remains active on the paralyzed side because its nerve supply comes from the intact superior laryngeal nerve and it operates exclusively to change vocal frequency. The state of vocal fold as "recurrent laryngeal nerve paralysis" has been studied and described by Hirano below (1981, p.26):

Table 3
The State of Vocal Fold of RNL Paralysis

Location of Pathology	Glottal Incompetence	Symmetry	Uniformity	Layer Structure	Cover Mass Stiffness	Cover Mass Stiffness	Transition Mass Stiffness	Body Mass Stiffness	Interference
Muscle	Entire length (Marked)	Deteriorated	Maintained	Deteriorated	Normal	Normal	Normal	Decreased	None

(Dedo & Shipp, 1981)

Dedo and Shipp (1980) noted that the paralyzed vocal fold would vibrate like a normal vocal fold as it assumed a "wide paramedian position". According to the study of the paralyzed vocal cord in Japan, Kumakawa and his colleagues found that "the free margin of the paralyzed vocal cord shifted by the aerodynamic power along the direction during normal phonation and to the caudal during inspiratory phonation" through the unilateral RNL palsy, and "this displacement phenomenon of the paralyzed vocal cord atrophy" (Kumakawa et al., 1990, p. 61). Atrophy due to the paralysis is inevitable, which means sooner or later the hoarseness related to atrophy of the vocal fold increases in severity, and conflicts with what the surgery attempted to accomplish.

Recurrence of Spastic Dysphonia

Dedo and other researchers reported 10-15% of the patients who have undergone the RLN section have a return of spastic dysphonia within three years after the operation.

A Cause of Recurrence

1) "Chronic tightening of the vocal folds eventually results in a return of the inappropriate squeezing of the folds toward the midline with a subsequent manifestation of the strain-struggle phonation so characteristic of the early phases of spastic dysphonia" (Dedo & Shipp, 1980, p. 52).

2) The arytenoid cartilage may be in the wide paramedian positioning, a condition may result in a breathy post-operative voice. Dedo hypothesized that these individuals had strong muscle movement that interfered with vocal fold approximation.

Laryngologists need to check periodically as to whether or not the vocal fold is still paralyzed. After the examination if there is a recurrence of spasticity, two treatment options: 1) The

external branch of the superior laryngeal nerve may be sectioned for mild recurrence of spasticity. 2) In the presence of moderate to severe spasticity, substance may be removed from the paralyzed vocal fold. The purpose is to thin out the mass in order to pull the free edge back from the midline without scarring. Carbone dioxide laser via microdirect laryngoscopy is used for thinning the fold.

Treatment of Low-Intensity Voice

A weak, breathy voice is the prominent characteristic of the post-surgical voice of spastic dysphonia. In patients who might have and excessively wide glottis, Teflon injection into the paralyzed fold to assist in closing the gap is available. However, there is a risk of recurrence of spasticity again. For the paralyzed vocal cord, Kumakawa and his colleagues recommended the injection of silicon, too, to increase the vocal fold mass since it may become atrophied (1990).

Electrode Insertion into Recurrent Laryngeal Nerve

Friedman and his colleague (1987) introduced the treatment using the percutaneous insertion of the needle electrode into the region of the recurrent laryngeal nerve. They experimented it for the alternatives of the RLN resection. The result showed that the voice of patients with spastic dysphonia improved only when the needle electrode was transmitting current to the nerve. Therefore, Friedman and his colleagues suggested the implantation of nerve stimulators as the relatively safe and effective technique. They emphasized the merit of this inplantation as no dificit of recurrent laryngeal nerve.

Botulinum Toxin Injection

Botulinum toxin is a potent neurotoxin produced by the bacteria Clostridium botulinum found in one form of food poisoning. Type A, out of eight known strains, is the most potent and the only one that can be crystallized in stable form. When injected directly into the thyroarytenoid muscles in low doses, it binds firmly to muscle and is rapidly fixed at the terminal nerve fibers with little remaining toxin allowed to pass into the general circulation, according for its relative safety (Cohen and Thompson, 1987; Aronson, 1990, p. 338).

Physiological Effect of Botulinum Toxin

Botulinum toxin blocks the release of acetylcholine from the nerve ending, interfering with calcium metabolism, and in effect, denervating muscle fibers for month; because the motor end plate does not release acetylcholine, the muscle fibers cannot contract" (Aronson, 1990, p. 338). Approximately three months will take to reinnervate the muscle by developing new nerve sprout.

Procedure of Injection

The toxin is injected percutaneously through the cricothyroid membrane into the thyroarytenoid muscle by means of a syringe with a monopolar Teflon-coated hollow electromyographic recording needle, in order to locate the thyroarytenoid muscle before the toxin is injected into it (Aronson, 1990, p. 338).

The bilateral vocal fold injection is preferable as it requires considerably less toxin and causes a better voice than the unilateral vocal fold injection does (Blitzer et al., 1988).

Result of Botulinum Toxin Injection

Botulinum toxin takes effect within 24 to 72 hours: 1) initially the voice becomes very breathy or hoarse with some developing mild aspiration, 2) the voice becomes stronger and very fluent within the first 3 days (Blitzer et al., 1988, p.194). However, Aronson (1990) indicated that patients' voices responded differently due to differences in the severity of their spastic dysphonia, and to the amount of toxin that was used dependent on the individual patient's need. After several injections are performed, the optimum dose will be determined for the individual patient. The effect on the voice lasts from three to six months, and gradually, it declines. Therefore, reinjection is required when the effect is wearing off.

Advantages and Disadvantages of Botulinum Toxin Injection

The Botulinum toxin injection makes the voice quality as good or better than recurrent laryngeal nerve resection. The advantages of Botulinum toxin injection are: 1) the result is as good or better than recurrent laryngeal nerve resection with little discomfort, 2) being under general anesthesia is not necessary, 3) the toxin does not produce a permanent paralysis of a vocal fold, and 4) repeated injections produce similar or identical results. The disadvantage is the need of reinjection within three to six months. This Botox injection cannot cure spastic dysphonia like other treatments.

Discussion

This is the latest treatment for spastic dysphonia so far and the long-term effects of repeated injections have not yet been identified. Therefore, it is unknown for the longest effective period of repeated injection or how the immune system will work overtime (Aronson, 1990; Brin et al., 1978; Blitzer et al., 1988).

SUMMARY

Spastic dysphonia is a voice disorder in which the vocal folds are approximated so tightly that it is impossible to produce sustained vocal fold vibration. The etiology is idiopathic, however spastic dysphonia can be classified as psychogenic, neurologic, and idiopathic types. Differentiate diagnosis is crucial for the efficient treatment of spastic dysphonia because the name "spastic dysphonia" simply described a family of strained voice produced by adductor laryngospasm that arise from different etiologies. The cooperation of different disciplines is necessary and very important for thorough diagnosis.

Historically, various therapies have had little success in retreating spastic dysphonia. Recurrent laryngeal nerve resection has achieved a remarkable success in terms of improved voice and relief from effort to phonate. However, recurrence of spastic dysphonia after this resection is reported in many cases otherwise permanent unilateral RLN paralysis remains.

The latest treatment of spastic dysphonia is Botulinum toxin injection. Botulinum toxin

(Botox) is injected percutaneously into the thyroarytenoid muscle, which paralyze the vocal folds bilaterally. This paralysis lasts from three to six months and requires reinjection followed by the effect wearing off. Because this injection treatment is relatively new and the long-term effect has not been determined: optimum dose, reinjection frequency cycle, side effects, and immunologically mediated responses for long-term have to be identified in future.

References

Aminoff, M.J., Dedo, H.H., & Izdebski, K. (1978). Clinical aspects of spasmodic dysphonia. *Journal of Neurology Neurosurgery, and Psychiatry,* 41, 361-365.

Aronson, Arnold E. (1985). *Clinical Voice Disorders*. New York: Thieme Inc.

Aronson, Arnold E. (1990). Clinical Voice Disorders. New York: Thieme Inc.

Aronson, Arnold E., & DeSanto, L.W. (1983). Adductor spastic dysphonia: Three years after recurrent laryngeal nerve resection. *Laryngoscope,* 93, 1-8.

Aronson, Arnold E., Brown, J.R., Litin, M.E. & Pearson, J.S. (1968 a). Spastic dysphonia. I. Voice, neurologic and psychiatric aspects. *Journal of Speech and Hearing Disorders,* 33, 203-218.

Aronson, Arnold E., Brown, J.R., Litin, M.E. & Pearson, J.S. (1968 b). Spastic dysphonia. II. Comparison with essential (voice) tremor and other neurologic and psychogenic dysphonias. *Journal of Speech and Hearing Disorders,* 33, 219-231.

Arnold, G.E. (1959). Spastic dysphonia: I. Changing interpretations of a persistent affliction. *Logos,* 2, 3-14.

Blitzer, A., Lovelace, R.E., Brin, M.F., Fahn, S., & Fink, M.E. (1985). Electromyographic findings in focal laryngeal dystonia (spastic dysphonia). *Ann. Otol. Rhinol. Laryngol.,* 94, 591-594.

Blitzer, A., Brin, M.F., Fahn, S., & Lovelace, R.E. (1988). Localized injections of botulinum toxin for the treatment of focal laryngeal dystonia (spastic dysphonia). *Laryngoscope,* 98, 193-197.

Bloch, P. (1965). Neuro-psychiatric aspects of spastic dysphonia. *Folia Phoniatr,* 17, 301-364 .

Bocchino, J.V., & Tucker, H.M. (1978). Recurrent laryngeal nerve pathology in spasmodic dysphonia. *Laryngoscope,* 88, 1274-1280.

Brin, M.F., Fahn, S., Moskowitz, C. et al. (1987). Localized injections of botulinum toxin for the treatment of focal dystonia and hemifacial spasm. *Mov. Disord.,* 2, 237-254.

Boone, Daniel R. (1983) *The Voice and Voice Therapy*. (3rd ed.) N.J.: Prentice-Hall.

Brodnitz. F.S. (1976). Spastic dysphonia. *Ann. Otol. Rhinol. Laryngol.,* 85, 210-214.

Case, James L. (1984). *Clinical Management of Voice Disorders*. Rockvile, Maryland: An Aspen Publication.

Cohen, S.R. & Thompson, J.W. (1987). Use of botulinum toxin to lateralize true vocal cords: a biochemical method to relieve bilateral abductor vocal cord paralysis. *Laryngoscope,* 96, 534-541.

Cooper, M. (1980). Recovery from spastic dysphonia by direct voice rehabilitation. *Proceeding of the* 18*th congress of the International Association of Logopedics and Phoniatrics*. Washington, D.C. 597-584.

Critchley, M. (1939). Spastic dysphonia ("inspiratory speech"). *Brain: A Journal of Neurology, LXII,* 96-103.

Dedo, H.H., Townsend, J.J., & Izdebski, K. (1978). Current evidence for the organic etiology of spastic dysphonia. *Otolaryngology,* 86 (6), 875-880.

Dedo, H.H. (1976). Recurrent laryngeal nerve section for spastic dysphonia. *Ann. Otol. Rhinol. Laryngol.,* 85, 451-459.

Dedo H.H. & Shipp T. (1980). Spastic Dysphonia: *A Surgical and Voice Therapy Treatment Program*. Houston, Texas: College Hill Press.

Finitzo, T. & Freeman, F. (1989). Spasmodic dysphonia, whether and where: results of seven years of

research. *Journal of Speech and Hearing Disorders,* 32, 541-555.
Friedman et al. (1987). Treatment of spastic dysphonia without nerve section. *Ann. Otol. Rhinol. Laryngol.,* 96, 590-596.
Hall, J.W. & Jerger, J. (1976). Acoustic reflex characteristics in spastic dysphonia. *Archives of Otolaryngology,* 102, 411-415.
Hall, J.W. (1981). Central auditory function in spastic dysphonia. *American Journal of Otolaryngology,* 2 (3), 188-198.
Hannebert, P.E. (1972). L'Injection de Rethi dans certaines dysphonies par spasme des bandes ventriculares. *Acta oto-rhino-laryngologica Belgica,* 26, 801-805.
Hartman, D.E. (1980). Clinical investigations of abductor spastic dysphonia (intermittent breathydysphonia). *American Speech-Language-Hearing Association Convention, Detroit.*
Heaver, L. (1959). Spastic dysphonia : II. psychiatric considerations. *Logos* 2, 15-24.
Hirano, M. (1981). Structure of the vocal fold in normal and disease states anatomical and physical studies. *ASHA report, No.*11, 11-30.
Jankovic, J., & Ford, J. (1990). Blepharospasm and orofacial-cervical dystonia: clinical and pharmacological findings in 100 patients. *Annals of Neurology,* 13, 402-411.
Kiml, P.J. (1963). Le Classement des aphonies spastiques. *Folia Phoniat.,* 15, 269-277.
Kiml, P.J. (1963). Recherches experimentales de la dysphonie spastique. *Folia Phoniat.,* 17, 241-300.
Kumakawa, K. et al. (1990). Image analysis of the cantor of paralyzed vocal cords during phonation. *Nippon Jibiinkoka Gakkai Kaiho,* 93 (1), 61-70.
Luchsinger, R., & Arnold, G. E. (1965). *Voice Speech-Language: Clinical Communicology--Its Physiology and Pathology,* Belmont, CA: Wadsworth.
Mroun, F.B., Jacob, J.C., & Gowing, P. (1970). Dysphonia associated with cortical neoplasms. *Journal of Neurosurgery,* 32, 671-676.
McCall, G.N. (1973). Acoustic impedance measurement in the study of patients with spasmodic dysphonia. *Journal of Speech and Hearing Disorders,* 38 (2), 250-255.
Moses, P.J. (1964). *Voice of Neurosis,* N.Y.: Grune and Stratton.
Rethi A. (1952). Rolle des stylopharyngealen Muskelsystems im Krankheitsbild der Taschenbandstimme und der Dysphonia spastica. *Folia Phoniatrica,* 4, 201-216.
Robe, E., Brumlik, J., & Moore, P. (1960). A study of spastic dysphonia: neurologic and electroencephalographic abnormalities. *Laryngoscope,* 70 (3), 219-245.
Rollin, W.J. (1987). *The Psychology of Communication Disorders in Individuals and Their Families.* New Jersey: Prentice-Hall.
Schaefer, S.D., Finitzo-Hieber, T., Gerling, I.J., & Freeman, F.J. (1983). Brainstem conduction abnormalities in spasmodic dysphonia. *Ann. Otol. Rhinol. Laryngol.,* 92, 59-64.
Stemple, Joseph C. (1984). *Clinical voice pathology : theory and management.* Columbus, Ohio: Charles E. Merrill.
Traube, L. (1871). Spastische form der nervösen heiserkeit. *Gesammelte Beiträge zur Pathologie und Physiologie,* (2), Berlin : Hirschwald, 674-678.